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Determination of Mean Crevicular Fluid Volumes of Males and Females of Different Ages

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DETERMINATION OF MEAN CREVICULAR FLUID VOLUMES
OF MALES AND FEMALES OF DIFFERENT AGES

by

Gary W. Brankin

A Thesis Submitted to the Faculty of the Graduate School
of Loyola University of Chicago in Partial Fulfillment
of the Requirements for the Degree of
Master of Science

June

1977

DEDICATION

To my parents, William and Lorraine Brankin,
whose many years of guidance, understanding,
encouragement and many sacrifices made
possible my many dreams.

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Lastly, I wish to thank Mr. Martin Moran for his help in the processing of the statistics for this thesis.

VITA

The author, Gary William Brankin, is the son of William Joseph Brankin and Lorraine (Holmes) Brankin. He was born January 2, 1952, in Chicago, Illinois.

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CHAPTER I

INTRODUCTION AND STATEMENT OF PURPOSE

Gingival fluid is a fairly new topic in research related to dentistry. During the past ten to fifteen years much work has been published about gingival fluid in the following areas:

1. Composition of gingival fluid
2. Gingival microcirculation and flow
3. Permeability of the crevicular epithelium
4. Methods of collection

It has been found by measuring the intensity of gingival fluid flow, a quantitative evaluation of gingival tissue can be made. However, until recently no accurate quantitative measurement was available. A gingival crevicular fluid meter (Harco) has been developed which electronically measures minute fluid volumes by the reduction in capacitance between two sensors when in contact with a standardized filter paper strip containing the fluid to be measured. It is known that crevicular fluid volume increases with an increase in inflammation of the gingival tissue.

There has been much controversy on the existence of gingival fluid in uninflamed gingivae. Therefore, the purpose of this investigation is to determine the mean gingival crevicular fluid volume in normal uninflamed gingivae of males and females of different

age groups utilizing a crevicular fluid meter.

There is also a great deal of controversy on whether a correlation exists between the gingival pocket depth and the amount of gingival crevicular fluid volume. This study should shed some light on this discrepancy.

CHAPTER II

REVIEW OF THE LITERATURE

Serres (1817) referred to a fluid which passed into the gingival sulcus and was due to secretions from glands embedded in the gingival border against the tooth.

In G.V. Black's (1887) histologic evaluation of the periodontal membrane, he referred to a gingival fluid in his statement, "It seems to be through this space that the cells - so called salivary corpuscles - found under the free border of the gingivus, pass. These may be found at any time under the healthy gingivus, and their numbers are augmented with every irritation of the membrane. Indeed, close clinical examination makes it apparent that there is a slight secretion at this point that is not quite satisfactorily explained even yet by microscopic study of the part." He concluded by showing histologic evidence of a deep plexus of gingival glandular tissue clustered among the principle fibers of the periodontal membrane and encircling the root. This observation differs from Serres' work.

McCall (1924) has postulated gingival inflammation brought about by traumatic occlusion will stimulate acid production. He also believed this could lead to caries.

Stillman and McCall (1927) listed as one of the functions of the marginal gingiva "...being the cleansing of the enamel surface over which it lies by the serous secretion from its crevicular surface."

Boedecker (1931) showed through extensive histological work the absence of glands within the gingival tissue and states, "where this erroneous idea that glands exist in the gum originated is difficult to discover." This suggests the fluid emanating from the gingival crevice is not a secretion but an exudate, resulting from some inflammatory condition of the tissue. In 1933 Boedecker tested for acid production in the gingival crevice with the use of litmus paper to determine if dental erosion is caused by this phenomenon. His findings agree with McCall's theory (1924).

Miller (1938) defined the term 'crevicular exudate' as a "discharge from the gingival crevice" and noted changes in the crevicular exudate as a clinical sign of incipient periodontal disease. He stated, "Frequently, even with clinical signs of periodontal disease lacking, microscopic examination of the crevicular fluid reveals the presence of an unusual number of pus cells."

Waerhaug (1952) placed India ink into the healthy gingival crevices of dogs to demonstrate its dynamic nature. He found most of the India ink removed within a period of two hours following an increase in the flow of the gingival fluid. He believed these part-

icles were removed by a process of 'transudation'. He also concluded the flow fluid proceeded outward from the crevice more rapidly than the saliva is able to diffuse inward, therefore saliva would not be able to penetrate below the gingival margin to remove the India ink.

Waerhaug and Steen (1952) studied the environment within the gingival sulcus to determine the presence or absence of bacteria. In a second study, the pockets of dogs were introduced with a concentrated bacterial culture and the tissue reaction was examined histologically over a forty-eight hour period. Their findings indicated: (1) pockets without calculus or other deposits were sterile, (2) the presence of bacteria within the sulcus causes a necrosis of the epithelium and inflammation of the connective tissue with subsequent formation of an exudate. Within a period of forty-eight hours a bacteria free environment is restored, (3) "from all pockets there is a constant flow of cellular elements and tissue fluid." These results indicate gingival fluid provided a cleansing action, removing foreign particles which would be injurious to the sulcular epithelium.

Brill and Krasse (1958) examined the permeability of the gingival epithelium of dogs with a permanent dentition and clinically healthy gingiva. Injections of fluorescein sodium were administered intravenously. The occurrence of fluorescein in the gingival pockets

was recorded by means of filter paper strips. Two methods of collection were used: (1) extracrevicular, (2) intracrevicular. With the extracrevicular method filter paper was placed on the marginal and attached gingivae and mucosa within the vestibule. Only the filter paper closest to the opening of the gingival crevice showed the presence of fluorescein. The intracrevicular method was used to determine a more affirmative conclusion. Fluorescein on the strips was observed as early as thirty seconds after injection in 151 out of 167 crevices (90.4%). Filter paper strips were also placed on various other epithelial surfaces, but the presence of fluorescein was not detected. It was concluded gingival fluid was derived from serum and by its action was able to remove microorganisms and other minute particles from the gingival sulcus and thereby acted as a defense mechanism.

Brill and Bjorn (1959) correlated the results of the previous study to human beings. Twelve persons between the age of twenty and fifty-one with clinically healthy gingiva were studied. Each person was administered 2-4 grams of fluorescein sodium orally. The occurrence of fluorescein was examined two to three hours later by the use of filter paper strips placed within the gingival crevice for three minutes. Strips of filter paper were also placed on various other epithelial surfaces and on the surface of the major salivary glands. Their results showed the epithelial lining of the

gingival crevice and the nasal mucosa were permeable to fluorescein molecules. They also noted a correlation between the degree of inflammation and the quantity of fluorescein recovered on the filter paper strips placed in the gingival crevice. It was suggested the outgoing stream of fluid had a cleansing effect and may have prevented movement of material into the sulcus.

Brill (1959) studied the removal of particulate matter and bacteria from the gingival sulcus. Charcoal particles suspended in a solution of physiological saline were introduced into thirty-four clinically healthy gingival sulci of two dogs. Within a period of six to twenty-four minutes the majority of particles had been removed as evidenced by their deposition on filter paper placed over the opening of the crevice. Also, bacteria deposited within the gingival crevice showed a decrease in number with the passage of time. It was postulated the removal of bacteria and charcoal particles was caused by a flushing action of the tissue fluid and this fluid was serumal in origin and therefore could contain antibodies.

Brill and Krasse (1959) determined the effect of mechanical stimulation on flow of tissue fluid through the gingival pocket epithelium is increased initially and returns to normal within ten minutes. The soft tissues surrounding the teeth were stimulated by the following methods: (1) chewing, (2) mechanical stimulation,

(3) gingival massage. The effect of various kinds of stimulation was determined by the amount of fluorescein recovered on filter paper strips after the intravenous injection of the fluorescein sodium in dogs. The effects of the increased permeability were believed to be a result of the following factors: (1) a vascular bed mechanically stimulated results in arteriole dilation with an increased pressure and an increased permeability of the vessels allowing plasma to escape which, in turn, increases the quantity of interstitial fluid, (2) the ground substance of the connective tissue matrix could be described as a sol-gel system which exists as a gel in the resting state and sol when the tissue was mechanically stimulated.

Brill (1959) studied the effect of changes in capillary permeability influenced by histamine, mechanical stimulation, and inflammation by noting the flow of the gingival fluid. Evan's blue (vital dye which binds to plasma albumin in small concentrations and α -globulin in larger concentrations) was injected intravenously to aid in the determination. The results were summarized as follows:

1. "Short time mechanical stimulation and surgery recently performed produces a heavy outpour of Evan's blue from vessels into gingival pockets."
2. "Intravenously injected histamine in small concentrations has a similar effect; in large concentrations there is no appreciable effect."

3. "Chronic inflammation stimulated the escape of Evan's blue into gingival pockets appreciably, but compared with (1) the effect is small."
4. "Evan's blue passes into clinically healthy gingival pockets in minimum quantities."

It was again noted these plasma proteins contain antibodies and are antimicrobial in effect, therefore a health promoting effect could be attained by gingival massage.

Brill (1959) examined the effect of chewing on flow of tissue fluid in human gingival pockets. Mastication of the paraffin increased the flow of fluid into the gingival sulcus which he reasoned would have a positive effect on the gingival health. Brill also determined sulcular fluid, collected in resting as well as stimulated conditions, contains amino acids. Brill concluded, "When gingival structures are stimulated by chewing, the antimicrobial effect may be increased, because mechanical stimulation of the gingival vascular bed stimulated escape of fluid from the vessels and plasma contains several antimicrobial substances."

Brill and Bronnestam (1960) utilized immunoelectrophoresis to analyze fluid from human gingival sulci. At least seven protein components were identified including the β and γ globulins (showing antibodies may be present) and α_2 -macroglobulin (demonstrating serum components of high molecular weight can pass through

the capillary wall and connective tissue to reach the gingival sulcus). This added to the theory of the gingival fluid possessing antimicrobial properties.

Gavin and Collins (1961) investigated the occurrence of bacteria in the clinically normal gingival crevices. It was postulated a state of equilibrium existed between the entry of salivary microorganisms and the defense mechanisms eliminating them from the gingival crevice. Their results refuted Waerhaug and Steen's (1952) conclusion of the sterility of the healthy gingival crevice (free from calculus or deposits). They concluded, "by the described method the clinically healthy gingival crevice appears to contain microorganisms in the majority of cases."

Gavin and Collins (1961) also investigated the antimicrobial effect of fluid from the clinically healthy gingival crevice by placing filter paper strips with this fluid onto inoculated and uninoculated blood agar plates. They concluded, "The tissue fluid exudate from the clinically healthy gingival crevice has no appreciable bacteriostatic or bactericidal effect."

Gustaffson and Nilsson (1961) investigated the fibrinolytic activity of gingival fluid from clinically healthy gingivae. Filter paper strips analyzed indicated the presence of elements of the fibrinolytic system. The authors suggested, "fibrinolytic factors in the crevicular fluid might be of significance in counteracting the deposition of fibrin and other proteins at the junction between

the gingival epithelium and the tooth."

Löe (1961) studied the rate of epithelial cell turnover and the presence of leukocytes in clinically normal gingival crevices with an alcoholic solution of colophony (rosin) and used acrylic crowns to cover the gingival margin. Histological examination resulted in the following conclusions: (1) "mitotic figures along the entire length of the epithelial lining of the pocket and the desquamation of the surface cells support the view that the epithelial cuff is constantly renewed", (2) accumulation of neutrophilic leukocytes within the gingival sulcus indicated "that they migrate through the epithelial lining under physiological conditions," and (3) "there is a continuous transudation of tissue fluid into the clinically normal gingival pockets."

Harvey (1962) investigated the elimination of extraneous material of macroscopic size from the clinically healthy gingival crevice of dogs and humans. A silver alloy was used as the extraneous material. Harvey concluded the elimination of the alloy was due to the physiological flow of tissue fluid from the gingival crevice and it was suggested, "that the normal gingival crevice maintained its hygienic state by constant flushing with tissue fluid which was increased as a result of an acute inflammatory response to irritation."

Krasse and Egelberg (1962) determined the Na/K ratio of gingival fluid from clinically healthy and inflamed gingivae by using flame photometry. The results indicated the Na/K ratio of the fluid in both cases was lower than that of plasma, showing that intracellular potassium added to the extracellular fluid as it left the tissue into the gingival sulcus. Therefore, it was concluded, "that gingival pocket fluid cannot be regarded as a simple filtration product, but rather as an inflammatory exudate."

Brandtzaeg and Mann (1962) studied lysozyme activity of human gingival pocket fluid, serum, and saliva with varying degrees of inflammation. Lysozyme activity was measured by the change in turbidity of a cell suspension of killed and lyophilized Micrococcus lysodeikticus by using a spectrophotometer set at 625 mu. A wavelength of 625 mu was selected at which the specimen colors had the least influence of the results. They concluded, "there was an apparent trend toward higher activity of gingival fluid with increased severity of periodontal inflammation and destruction." This was not found to be the case in regard to serum and saliva. Also, there was no correlation of the lysozyme activity of the three fluids. It was suggested lysozyme within the gingival fluid is primarily of local origin, possibly from leukocytes. The effects of lysozyme were discussed and hypothesized as follows:

I. Advantages

1. Destruction of gram + bacteria by breaking down their cell walls.
2. Lysozyme can agglutinate bacteria for efficient phagocytosis.
3. Lysozyme within gingival pocket can reduce numbers of bacteria and their virulence.

II. Adverse Effects

1. Lysozyme can lyse some gram - bacteria releasing endotoxins which are injurious to the local tissue.
2. Lysis of bacteria release endocellular bacterial enzymes:
 - A. hyaluronidase
 - B. chondroitin sulfatase
 - C. gelatinase
 - D. collagenase

They suggested, "all of these endocellular toxins and enzymes may play a role in the etiology or perpetuation of periodontal disease."

Egelberg (1963) compared the cellular content of gingival pocket fluid from clinically healthy gingivae to that of fluid from chronically inflamed gingivae. It was found both samples contained epithelial cells, polymorphonuclear leucocytes, lymphocytes,

and bacteria, but fluid from the chronically inflamed gingivae contained many more bacteria and disintegrated leucocytes and some red blood cells. This agrees with the concept fluid in healthy pockets is a inflammatory exudate.

Mann (1963) collected gingival fluid from human subjects to determine if correlation exists between gingivitis, pocket depth, and the amount of exudate from the gingival crevice. The gingival fluid was collected with filter paper strips two hours after fluorescein sodium was administered orally. His results indicated:

(1) relating pocket depth and flow per unit area there was no significant correlation ($r = .232$), (2) comparing the gingival score (Parfitt Index) and flow per unit area there is significant positive correlation ($r = .473$), (3) whenever gingivitis was not present flow was minimal or absent. He concluded, "these variables indicated that inflammation was the main factor contributing to the rate of fluid flow."

Egelberg (1964) determined if gingival fluid exudate measurements could be used to evaluate the inflammatory state of the gingivae by performing three experiments.

1. Egelberg measured the exudate from three different regions of each tooth to determine what amounts of exudate can be obtained from clinically healthy gingivae and if variation existed in the amount obtained from each area. He found

the amounts to be at constantly low levels and the mesial area to have larger amounts of exudate compared to the buccal and palatal areas. He stated this is due to gingival papillae having a higher degree of inflammation and measurements should be taken only in the papillary regions.

2. In his second part of the investigation he found a positive correlation ($r = .90$) between the recorded amounts of exudate and the clinical scores for gingival inflammation (inflammation was scored on a scale from 0 - 3; 0 related to healthy and 3 marked chronic inflammation).
3. In his third part of the investigation he also found a positive correlation between amounts of exudate and the degree of inflammation of the gingivae assessed from histological preparation (measurements of inflammatory cell infiltration). Egelberg concluded, "that gingival exudate measurements can be considered a method which fulfills rather great demands in regard to objectivity and sensitivity and that this method increased possibilities to study the effect of various factors on inflammation of the gingivae have probably been achieved." He also concludes measurements of fluid are able to relate small changes in the level of inflammation.

Weinstein and Mandel (1964) published a review of the literature on the origin of gingival crevicular fluid. In their opinion, the best explanation was to call it a specifically altered transudation from serum modified by the cells of the crevicular area and is explained as follows: (1) the epithelial cells shed add their intracellular contents to the transudation, (2) the cells may exhibit ion transport altering the nature of the fluid, (3) the cells may add cytocellular fluid by exchange through the micropores of the cell wall.

Löe and Holm-Pedersen (1965) studied the absence and presence of fluid from normal and inflamed gingivae by the use of filter paper strips using an extracrevicular technique (strip placed next to the tooth surface, the gingival margin, and the attached gingivae) and an intracrevicular technique (strip placed at the entrance of the gingival crevice). Their conclusions were: (1) "Crevices of normal human gingiva do not exhibit flow of fluid, and mechanical stimulation of the periodontium does not produce fluid from such crevices" (contrary to the findings of Brill-1959, 1962 and Brill and Krasse-1959). (2) "Inflamed gingiva shows the presence of fluid, the amount of which varies according to the severity of the inflammation. The flow starts before structural changes can be ascertained at the clinical level and persists some time after clinical inflammation has subsided." (3) "Gingival fluid is an

inflammatory exudate and that the absence or presence of fluid may represent the definite clinical criterion in the refined distinction between normal and inflamed gingiva."

Brandtzaeg (1965) studied the presence of albumin, fibrinogen, IgG, IgA, and IgM by immunoelectrophoresis within crevicular fluid. He concluded plasma was the principle source of these proteins because (1) they possessed antigenic properties similar to plasma proteins, (2) they migrated similar to plasma proteins upon electrophoresis, and (3) their concentrations were equivalent to those of plasma. Therefore the author states gingival fluid is an exudate rather than a transudate.

Bader and Goldhaber (1966) administered therapeutic doses of tetracycline intravenously into female mongrel dogs and determined the presence of the antibiotic within the gingival crevice and saliva. The antibiotic was detected in the gingival fluid by U.V. light within thirty seconds after administration. The antibiotic was not found in secretions of the major salivary glands or unstimulated saliva. Therefore it was postulated the major source of systemically administered tetracycline is through the gingival crevice. These findings agree with those of Brill and Krasse (1958).

Sueda, Cimasoni, and Held (1966) compared the histochemical properties of gingival fluid to serum and saliva in five young adults with clinically healthy gingivae. They utilized specific

stains and found gingival fluid to contain proteins, lipids and polysaccharides bound to proteins (muco-, glyco-, or lipoproteins) which was similar to serum and weak or absent in saliva. Their results confirmed Cimasoni's (1966) hypothesis that a mucopolysaccharide substance identified by Toto and Sicher (1964), resistant to hyaluronidase, found between the enamel and the crevicular epithelium could represent a condensation of gingival fluid.

Stallard (1967) studied the microcirculation of the periodontium of squirrel monkeys by injecting plastic microspheres (15 ± 5 μ m) into the external carotid artery. The average diameter beneath the attached gingivae was 16.8 μ m. The vascular arrangement was found to change in areas of chronic inflammation in terms of vessel size and capillary permeability. It was concluded, "A definite correlation exists between the integrity of the microvasculature and sulcular epithelium and the presence of gingival crevicular fluid," and "the inflammatory reaction, with its characteristic vascular alterations, is a physiological defense mechanism; however, it appears that in the case of periodontal disease the inflammatory response eventually becomes pathologic."

Weinstein et al. (1967) used chemical, immunochemical, electrophoretic and electron microscopic techniques to demonstrate and characterize the properties of gingival fluid in clinically healthy gingivae. They conclude gingival fluid does exist in

clinically healthy gingivae and normal complement of organic components were found, but at different concentrations than in serum. Therefore "gingival fluid cannot be considered purely a transudate of serum," but "it is a transudate of serum modified by the cells of the sulcular area."

Kleinberg and Hall (1968) measured the pH and depth of gingival crevices in eighteen different regions of the mouths of fasting humans with clinically healthy gingivae. The pH measured with antimony micro-electrodes which had marks on the insulation to determine crevice depth simultaneously. They found the crevice pH for each region examined to be higher than the supragingival plaque pH in the same region by an average of 0.64 units higher. They also found the pH of the mandibular crevices was higher than pH of corresponding maxillary crevices and interproximal crevices higher in pH than lingual or labial-buccal crevices. The authors suggest, "saliva is also a determinant of the crevice pH." The more saliva exposed to a crevice the higher the pH. The authors postulate dietary substrates (carbohydrates) have difficulty reaching the crevices, therefore rate of base formation should be higher than acid formation in the crevices. They also found a high degree of correlation between the supragingival plaque pH and the crevice depth suggesting, "intraoral factors favouring higher plaque pH levels also favour deeper crevices and are probably responsible

for the variation in the susceptibility to periodontal disease."

They also found "up to a crevice depth of approximately 0.7 mm, the pH increased whereas above this depth the pH decreased." They concluded, "periodontal disease and caries are two interrelated pathological conditions that reflect the metabolism of the plaque and crevice microfloras each of which is associated with different regions of the pH scale."

Orban and Stallard (1969) studied the correlation of gingival health and amount of crevicular fluid flow. Gingival health was determined by utilizing gingivitis and plaque scores according to Ramfjord's criteria and Green and Vermillion's oral hygiene index. Biopsies were taken and evaluated for inflammatory infiltrate and scored on a 0 - 10 scale for evaluation. The amount of gingival fluid was measured by placing filter paper strips into the pockets for three minutes and treating with the conventional ninhydrin technique. They found the crevicular fluid scores were not correlated to any of the biopsy scores and correlation did exist between plaque scores and biopsy scores. Therefore it was concluded, "a better indication of the inflammatory status of the gingival tissue, as revealed by biopsies, is the evaluation or measurement of dental plaque."

Oliver, Holm-Pedersen and L  e (1969) studied the relationship between a gingival index (L  e and Silness, 1963), the exudate measure-

ment and the microscopic appearance. Exudate measurements were done by placing filter paper strips intracrevicularly for three minutes and then treating with the ninhydrin staining technique. They concluded when there is no clinical evidence of gingival inflammation the presence of exudate is not detectable in the majority of cases. Also, it was concluded, "the statistical analysis of the data demonstrated a close relationship between the Gingival Index scores and the gingival exudate measurements."

Turner et al. (1969) established a perfusion technique to anatomically visualize the pattern of microcirculation of the periodontium and teeth in dogs. This involves injecting a one percent solution of Patent Blue V in one carotid artery and filtered Pelikan carbon black suspension in the other carotid artery. They concluded: (1) one percent Patent Blue V "passes quickly through the epithelial attachment and the sulcular epithelium into the gingival sulcus," (2) "the Patent Blue V perfusion offers some evidence that a dye labelled-transudate passed from the capillary loops of the peripheral vascular plexus of the gingiva and entered the intercellular matrix, since there is evidence of tissue staining," and (3) Pelikan carbon black suspension (particle size 200 - 500 Å) "does not pass through the intact capillary wall."

Rudin, Overdick, and Rateitschak (1970) determined the correlation between sulcus fluid rate and clinical and histological inflammation of the marginal gingiva in humans. Sulcus fluid was collected

using L  e's technique and the condition of the gingiva was scored using the M-Index. Results were as follows: (1) healthy marginal gingivae contained very slight amounts of crevicular fluid, (2) the rate of fluid flow increased as the intensity of the inflammatory process increased. It was concluded crevicular fluid rate can be equated with the severity of marginal gingivitis.

Renggli and Regolati (1972) determined the absence or presence of leukocytes within the gingival crevice and its relation to the amount of gingival fluid collected in humans with clinically healthy gingivae. They concluded, (1) all crevices contained leukocytes, (2) "leukocyte count varies considerably among subjects, teeth, and days," (3) "no correlation exists between flow rate and number of leukocytes."

Egelberg and Attstrom (1973) compared the results of gingival fluid measurements sampled with the orifice method and intracrevicular method, when changes in the degree of gingival inflammation were induced in humans and dogs. They concluded, "the techniques seem to be comparable for evaluation of intraindividual changes of gingival inflammation."

Alfano (1974) published an extensive review on the origin of gingival fluid and proposed a theory which would explain the opposing views on the origin of the fluid. The two views on the origin of gingival fluid were (1) physiological transudate, and (2) pathological inflammatory exudate. Alfano's theory which explained the

above controversies was gingival fluid may arise by two distinct mechanisms: (1) "the generation of a standing osmotic gradient, and (2) the initiation of classical inflammation." Therefore, it was concluded, "gingival fluid may progress, at different times or in various areas of the mouth, from an initial osmotically modulated exudate to a secondary inflammatory exudate, with consequent alterations in its composition."

Borden et al. (1974) compared extracrevicular and intracrevicular techniques by measuring the human crevicular fluid flow using a fluid meter (Harco Electronics LTD., Winnipeg, Canada). They also determined if there was any correlation between gingival fluid flow and the Gingival Index (Löe and Silness-1963) and pocket depth. Their conclusions were (1) "repeated intracrevicular measurements gave similar gingival crevicular fluid flow rates," (2) the three second intracrevicular measurement was more sensitive than the extracrevicular measurement, (3) the recommended technique for measuring gingival crevicular flow rate using the Harco GCF meter is to initially empty the crevicular pool with a paper strip and thirty seconds later place a fresh strip intracrevicularly for three seconds, (4) "measuring gingival crevicular fluid (GCF) flow appears to be a sensitive technique for detecting early gingival inflammation," (5) "results indicated that the extracrevicular technique is unsatisfactory for slightly inflamed gingival crevices,

since GCF may have difficulty flowing out of such crevices."

Shern et al. (1974) compared the ninhydrin staining method and the use of the crevicular fluid meter (Harco Electronics LTD., Winnipeg, Canada) for quantifying crevicular fluid flow in humans. They concluded, "precision, accuracy and reliability of measuring crevicular flow proved greater using a flow meter than using the ninhydrin dye method."

Golub et al. (1974) determined whether collagenolytic activity could be detected in human crevicular fluid and if this activity was related to gingival disease. Results indicated (1) no collagenolytic activity from gingiva with a mean gingival index of $1.07 \pm .13$, a mean pocket depth of 2.38 ± 0.26 mm, and a mean GCF volume (Harco) of 0.16 ± 0.02 ul., (2) collagenolytic activity from gingiva with a mean gingival index of 1.44 ± 0.21 , a mean pocket depth of 2.61 ± 0.26 mm, and a mean GCF volume of 0.26 ± 0.05 ul. Therefore, it was concluded, "collagenolytic activity in human GCF tends to be associated with gingiva exhibiting increased inflammation."

Lie and Selvig (1975) tested the hypothesis the dental cuticle is formed by adsorption of material from serum and tissue fluid which escaped through the junctional epithelium as gingival exudate. They did this by comparing the histochemical staining reactions and morphology of the dental cuticle to those of cuticular structures experimentally incubated on tooth surfaces in serum. Their obser-

vations were: "enamel and denuded cementum and dentin surfaces which are exposed to serum or related fluids at 37°C will adsorb stainable material from this fluid by a purely physical-chemical process." They stated the dental cuticle reflects "the ubiquitous presence of inflammation of the gingiva and exudation through the junctional epithelium."

Holm-Pedersen et al. (1975) produced experimental gingivitis in young and elderly individuals by instructing them to abstain from normal oral hygiene procedures for a period of twenty-one days. Their results indicated: (1) "the discrepancy in inflammatory response between young and old indicates the development of gingival inflammation may vary between young and old individuals when challenged by bacterial plaque." (2) both groups developed clinical gingivitis during the period of oral hygiene abstention. (3) "the flow of exudate increased throughout the period of oral hygiene abstention." (4) "the development of gingival inflammation during the oral hygiene abstention period was more rapid and severe in elderly individuals." (5) "Measurements of crevicular exudate revealed the difference between young and old individuals more markedly than did the Gingival Index." (6) tissue recovery was slower in the elderly individuals following re-initiation of oral hygiene procedures than the younger individuals. (7) "alterations in the immune response to dental plaque antigens with ageing may offer an explanation for the more severe effect in the elderly."

Makinen and Hyyppa (1975) studied the origin of arginine aminopeptidases in human gingival fluid (in varying clinical states), whole saliva, plaque, serum and erythrocytes. They concluded, (1) "the severity of inflammation increased the relative proportion of the enzymes," (2) "clinically healthy gingivae produced fluid containing the same enzymes as fluid derived from diseased tissue," (3) "an enzyme resembling aminopeptidase B was present in all gingival fluids." It was suggested the source of enzymes were as follows: (1) plaque, (2) serum, (3) erythrocytes.

Squier and Johnson (1975) discussed the permeability of gingival and sulcular epithelium and stated enzymes, toxins and antigens from plaque invade the host tissue and produce inflammation and destruction of tissue supporting the teeth and this reaction is primarily due to an immunological response. The external aspect of the gingiva didn't demonstrate penetration through the epithelium, but the sulcular epithelium was found to be permeable to substances up to a molecular weight of 1.0×10^6 and the intercellular space in the junctional epithelium demonstrated a substantially greater permeability than in the sulcular epithelium or external epithelium. He stated substances which are lipid soluble are more likely to pass along membranes and these are limited by the presence of a keratin layer and substances pass across skin and oral mucosa by simple diffusion and obey Fick's Law: "the rate is directly proportional to the concentration of the penetrant." They hypothesized, crevicular

fluid leaves the tissue at the junction of the oral sulcular epithelium and junctional epithelium and concluded, "even in the intensely studied gingival area, we have little information concerning the intrinsic permeability of this tissue."

Golub and Kleinberg (1976) published a review of the literature on gingival crevicular fluid and pointed out it can be used as an aid to diagnose the periodontal patient. They stated whether the fluid is altered serum transudate or inflammatory exudate is academic, "since the important point is whether or not the epithelial cells lining the crevicular tissue can modify the composition of the GCF flowing from clinically-normal or near-normal tissues." They agreed with Alfano's theory (1974) of an osmotic gradient generated by the accumulation of macromolecular by-products of bacterial metabolism and degradation in the gingival crevice, or in other words, the metabolic activity of the subgingival plaque would modulate the initial flow of GCF and they added to this hypothesis these by-products also increase flow by "directly affecting the crevicular epithelium and the cells of the underlying connective tissue rather than simply increasing the osmotic pressure." They discussed the progression of periodontal disease and stated, (1) ammonia could be an initiator in the gingivitis process, (2) the high level of plaque ammonia is the main cause of the alkaline pH of plaque (the main source of ammonia being salivary urea), (3) ammonia penetrates cell membranes easily, (4) ammonia influences intracellular

processes such as glycolysis, (5) ammonia is a cell irritant, (6) ammonia inhibits wound healing, (7) urea is important in progression of the disease by supplying plaque bacteria with substrate to produce more ammonia. They stated, "monitoring GCF flow is a sensitive indicator of alterations in gingival status and the intra-crevicular method is the more sensitive procedure." They discussed and concluded the GCF flow is a measure which helps the clinician and patient advantageously in the following ways: (1) measure of the healing following gingival surgery, (2) measure of the effectiveness of oral hygiene, (3) measure of the effectiveness of periodontal therapy, (4) detect subclinical gingival pathology, (5) "monitoring GCF for various components could provide the dentist with a valuable means of easily screening patients for systemic disease."

Golub et al. (1976) studied the collagenolytic activity of GCF from normal and inflamed gingival flap tissues overlying partially erupted lower third molars with the use of collagen gels, treatment with NaSCN, and disc gel electrophoresis. They concluded, (1) "collagenase activity was observed only in fluids originating from tissues with pericoronitis, (2) fluid from normal looking tissue exhibited collagenase activity with NaSCN treatment only if there was a history of pericoronitis, (3) collagenase activity originated from gingival tissue and not from bacterial origin,

because of the following: (1) "the activity of the untreated fluid seemed to parallel the activity of the adjacent gingival tissue," (2) NaSCN affected the fluid collagenase the same as it affected gingival tissue collagenase, (3) "the disc gel electrophoretic pattern of the breakdown products of collagen produced by the GCF enzyme was similar to that produced by gingival tissue collagenase and differed from that produced by a bacterial collagenase reported to exist in human gingival fluid."

Suppipat (1976) evaluated the HAR-600 Gingival Crevice Fluid Meter (Harco Electronics LTD., Winnipeg, Canada) and tested its usefulness in clinical trials. He determined (1) measuring gingival crevicular fluid with use of the HAR-600 is sensitive and valuable to evaluate the marginal gingiva, (2) meter readings may vary with different strip location in machine, viscosity of fluid, and climate. Clinically he concluded (1) none or very little fluid was collected from clinically healthy gingiva with the intracrevicular technique, (2) gingival fluid flow was related more to gingival inflammation than to pocket depth.

CHAPTER III

MATERIALS AND METHODS

Patients participating in this project were selected from those being treated at Loyola University Dental School Clinic. A consent form (see Appendix A) describing the procedure was signed by each patient and a brief medical and dental history completed (none of the patients had a history of systemic disease). Thirty-two patients between the ages of six and sixty-eight with a Gingival Index of 0 (after L  e and Silness - 1963) were selected and the depth of the sulcus measured (see figure 1) and recorded (see Table 1).

Gingival Index - L  e and Silness

<u>Score</u>	<u>Clinical Findings</u>
0	Absence of Inflammation.
1	Mild Inflammation: Slight change in color and little change in texture.
2	Moderate Inflammation: Moderate glazing, redness, edema and hypertrophy. Bleeding on pressure.
3	Severe Inflammation: Marked redness and hypertrophy; tendency to spontaneous bleeding; ulceration.

Teeth selected for measuring gingival crevicular fluid flow were maxillary right canine #6, lateral incisor #7, and central incisor #8.

Two measurements, mesial buccal and distal buccal near the interdental papillae area of each tooth were taken; therefore a total of six fluid volume measurements were taken on each patient giving a total of one hundred ninety two fluid volume measurements for the entire study (see Table 1). All measurements were taken by the same investigator. The collection technique followed the method suggested by the manufacturer (Harco Electronics Limited, Winnipeg, Canada) and the findings of Borden (1974) in regard to the use of this particular instrument (see figure 2). The collection technique was as follows:

1. Region to be examined was dried and isolated with sterile cotton rolls.
2. A sterile dry filter paper strip (Periopaper; Harco, 1.5 mm x 13 mm; see figure 3) was placed at the entrance to the gingival sulcus orifice (after L e and Holm-Pedersen-1965; see figures 4 and 5) for three seconds to empty the crevicular pool. This filter strip was removed and discarded.
3. After a twenty-seven second interval, another sterile dry filter paper strip was placed at the sulcus orifice for three seconds. The total elapsed time was thirty seconds.

4. The filter paper strip was immediately placed between the recording sensors so the entire moistened area of the filter paper strip was in contact with the sensors (the filter paper strip was inserted to the line marked on each filter strip; see figures 3, 6, and 7).
5. The digital read-out value on the Periotron[®] rose to a maximum and held at that value. The highest numerical reading was recorded. Then this relative numerical value can be converted to fluid volume (microliters) by dividing the reading by 200 (e.g. a digital reading of 20 = 0.10 ul.).
6. After each measurement, the sensors were dried with a sterile cotton roll.

Information from each data sheet was compiled into a single table (see Table 1) dividing the patients into the following age groups: 6 - 12, 13 - 20, 21 - 30, 31 - 45, 46 - 60, 60+.

Crevicular fluid volumes were subjected to analysis of variance to determine if a statistical significant difference existed between the different age groups (see Tables 2 - 8). A correlation-regression was performed to determine if correlation existed between the gingival pocket depth and the amount of gingival crevicular fluid flow (see Table 9).



Figure 1. Measuring Sulcus Depth Utilizing a Perioprobe.

Figure 2. Periostar® (Clinical GCF Meter)



Figure 2. Periotron[®] (Clinical GCF Meter)



Figure 3. Sterile Filter Paper Strip.

Figure 4. Vaginal Cervical Fluid Collection from a Patient.

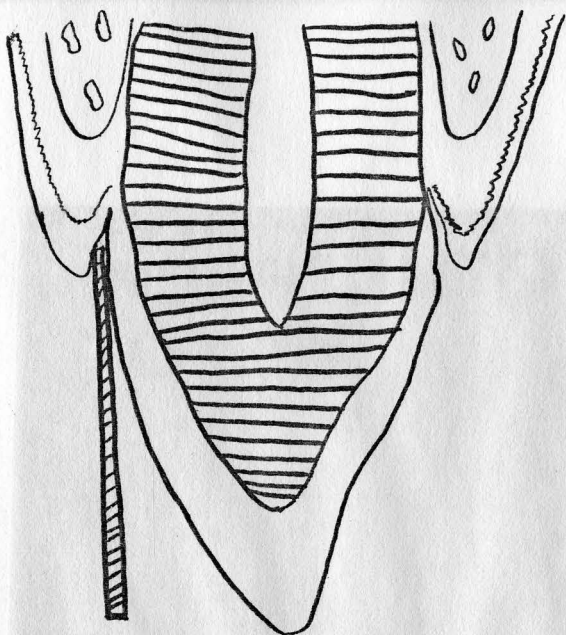


Figure 4. Diagrammatic representation of filter paper strip insertion.



Figure 5. Gingival Crevicular Fluid Collection from a Patient.

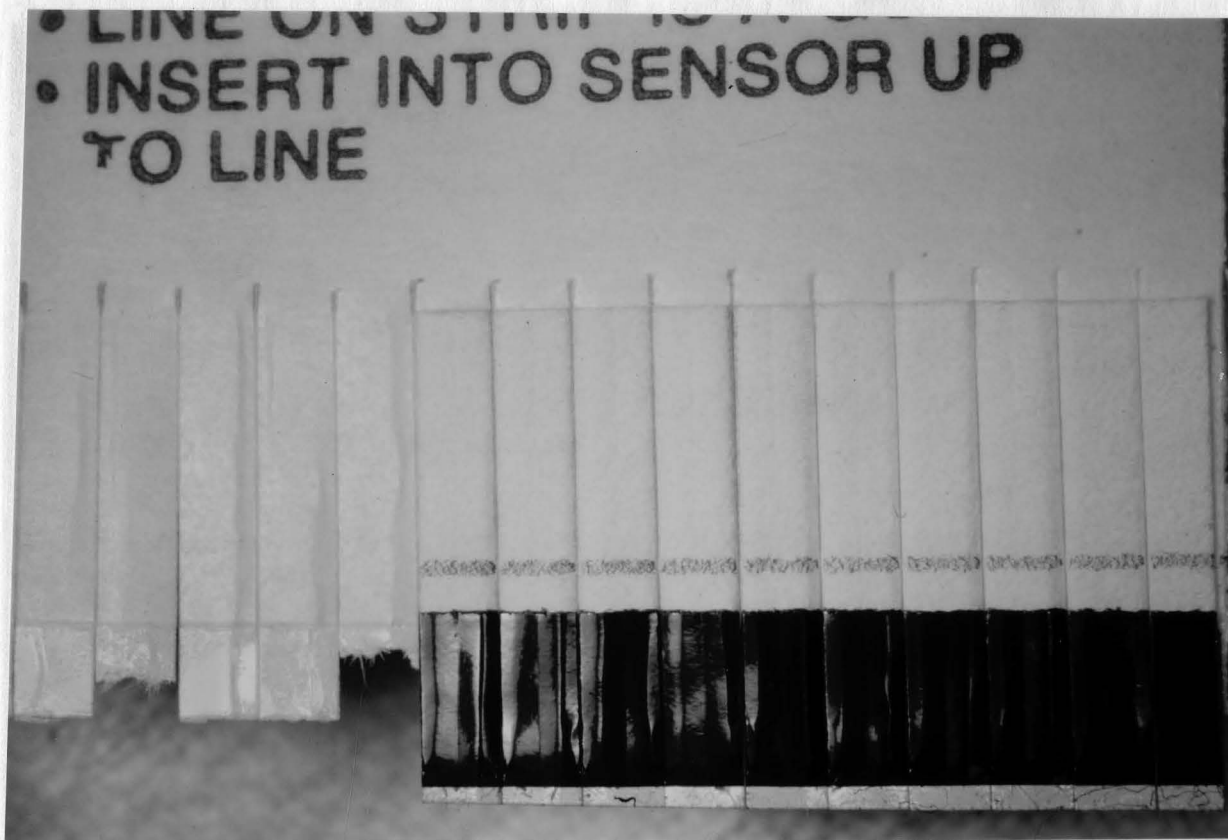


Figure 6. Holding Card for Filter Paper Strips.



Figure 7. Placing Filter Paper Strip Between the Recording Sensors of the GCF Meter.

CHAPTER IV

EXPERIMENTAL RESULTS

Table one consists of the compiled data from individual data sheets (see Appendix A) listing the patients number, age, sex, the recorded values for crevicular fluid volume and pocket depth for their respective teeth, total fluid volume and average fluid volume (instrumental readings and microliters).

Tables two through seven represents analysis of variance tables using various variables as listed on each table. Also, each table lists the F probability.

Table eight is the analysis of variance table for the total gingival crevicular fluid volumes showing an F ratio of 2.80 indicating a significant ($P < .05$) difference between the total crevicular fluid volumes of different age groups. Using a K-distribution, the following was determined:

1. The mean gingival crevicular fluid volume of age groups (6-12), (13-20), (46-60) and (60+) was not significantly different.
2. The mean gingival crevicular fluid volume of the above four groups were significantly higher than age groups (21-30) and (31-45).

Table nine summarizes the mean values of pocket depth and GCF meter readings.

Table ten summarizes the distribution of males and females within the different age groups.

Table eleven lists the coefficient of correlation and its significance to show there was no correlation between the depth of the gingival sulcus and the amount of gingival crevicular fluid flow.

Patient Number	Age	Sex	[Fluid Volume (Meter Reading)]		Pocket Depth (mm)				Total Fluid Volume	Average Fluid Volume	Average ul
			#6 Distal	#6 Mesial	#7 Distal	#7 Mesial	#8 Distal	#8 Mesial			
1	11	F	9/2	8/2	7/2	7/3	5/3	5/2	41	6.83	.034
2	11	M	6/2	6/2	6/2	4/2	4/3	4/2	30	5.00	.025
3	10	F	10/3	12/3	10/2	10/2	10/2	9/2	61	10.16	.050
4	9	M	10/2	10/2	7/3	6/3	6/2	6/2	45	7.50	.038
5	12	M	8/2	7/2	5/3	5/3	5/2	5/2	35	5.83	.029
6	6	M	6/2	5/2	7/1	5/1	3/2	4/2	30	5.00	.025
7	10	M	10/2	8/2	4/2	12/2	8/2	6/2	48	8.00	.040
8	15	M	6/2	10/2	8/2	6/3	10/3	7/3	47	7.83	.039
9	16	F	14/2	8/2	7/2	4/2	2/2	4/2	39	6.50	.033
10	14	F	8/2	6/2	5/2	5/3	5/3	4/2	33	5.50	.028

Table 1
Compiled Data

Patient Number	Age	Sex	[Fluid Volume (Meter Reading)]		Pocket Depth (mm)				Total Fluid Volume	Average Fluid Volume	Average ul
			#6 Distal	#6 Mesial	#7 Distal	#7 Mesial	#8 Distal	#8 Mesial			
11	19	F	9/2	9/2	8/3	6/3	7/2	7/2	46	7.66	.038
12	20	F	9/2	8/2	7/2	7/2	7/2	7/2	45	7.50	.038
13	25	M	7/2	5/3	5/3	6/3	7/2	4/3	34	5.66	.028
14	21	M	4/3	4/3	6/3	6/3	6/2	6/2	32	5.33	.027
15	27	F	9/2	8/2	8/3	10/2	8/2	4/2	47	7.83	.039
16	26	F	6/3	5/3	6/2	6/2	7/2	8/2	38	6.33	.032
17	22	F	6/2	7/2	1/2	2/2	0/2	0/2	16	2.67	.013
18	26	M	2/2	2/2	6/2	6/2	1/2	1/2	18	3.00	.015
19	25	M	3/3	4/3	4/2	3/2	2/2	2/2	18	3.00	.015
20	40	F	4/2	2/2	0/2	0/2	0/2	0/2	6	1.00	.005

Table 1 (Continued)
Compiled Data

Patient Number	Age	Sex	Fluid Volume (Meter Reading)		Pocket Depth (mm)				Total Fluid Volume	Average Fluid Volume	Average ul
			#6 Distal	#6 Mesial	#7 Distal	#7 Mesial	#8 Distal	#8 Mesial			
21	40	M	8/3	10/3	4/3	3/3	4/3	2/3	31	5.17	.026
22	42	M	4/3	4/3	4/2	2/2	1/2	3/2	18	3.00	.015
23	40	F	6/3	5/2	3/2	3/2	4/3	4/2	25	4.16	.021
24	38	F	8/3	7/3	6/2	6/2	5/2	6/2	38	6.33	.032
25	49	M	10/2	9/2	10/2	10/2	7/3	7/3	53	8.83	.044
26	59	F	10/2	12/2	7/2	8/2	10/2	12/2	59	9.83	.049
27	54	F	8/2	7/2	7/2	8/2	6/2	6/2	42	7.00	.035
28	57	F	3/3	4/3	3/3	5/3	4/2	4/2	23	3.83	.019
29	51	F	6/2	6/2	6/2	5/2	4/2	4/2	31	5.17	.026
30	63	F	10/2	9/2	6/3	4/2	4/2	4/2	37	6.17	.031

Table 1 (Continued)
Compiled Data

Patient Number	Age	Sex	[Fluid Volume (Meter Reading)]		/		Pocket Depth (mm)		Total Fluid Volume	Average Fluid Volume	Average ul
			#6 Distal	#6 Mesial	#7 Distal	#7 Mesial	#8 Distal	#8 Mesial			
31	65	F	8/2	9/2	7/2	5/2	5/2	5/2	39	6.50	.033
32	68	M	14/2	8/2	10/2	7/2	8/2	6/2	53	8.83	.044

Table 1 (continued)
Compiled Data

Table 2

Analysis of Variance Table

(using volume measurements of
the distal aspect of tooth #6
as the variable)

<u>VARIANCE SOURCE</u>	<u>DF</u>	<u>SUM OF SQUARES</u>	<u>MEAN SQUARES</u>	<u>F RATIO</u>	<u>F PROB.</u>
Between	5	96.15	19.23	3.12	0.02
Within	26	159.81	6.14		
Total	31	255.96			

Table 3

Analysis of Variance Table

(using volume measurements of
the mesial aspect of tooth #6
as the variable)

<u>VARIANCE SOURCE</u>	<u>DF</u>	<u>SUM OF SQUARES</u>	<u>MEAN SQUARES</u>	<u>F RATIO</u>	<u>F PROB.</u>
Between	5	64.24	12.84	2.44	0.06
Within	26	136.72	5.25		
Total	31	200.96			

Table 4

Analysis of Variance Table

(using volume measurements of
the distal aspect of tooth #7
as the variable)

<u>VARIANCE SOURCE</u>	<u>DF</u>	<u>SUM OF SQUARES</u>	<u>MEAN SQUARES</u>	<u>F RATIO</u>	<u>F PROB.</u>
Between	5	56.23	11.24	2.66	0.04
Within	26	109.63	4.21		
Total	31	165.87			

Table 5

Analysis of Variance Table

(using volume measurements of
the mesial aspect of tooth #7
as the variable)

<u>VARIANCE SOURCE</u>	<u>DF</u>	<u>SUM OF SQUARES</u>	<u>MEAN SQUARES</u>	<u>F RATIO</u>	<u>F PROB.</u>
Between	5	65.69	13.13	2.45	0.05
Within	26	139.18	5.35		
Total	31	204.87			

Table 6

Analysis of Variance Table

(using volume measurements of
the distal aspect of tooth #8
as the variable)

<u>VARIANCE SOURCE</u>	<u>DF</u>	<u>SUM OF SQUARES</u>	<u>MEAN SQUARES</u>	<u>F RATIO</u>	<u>F PROB.</u>
Between	5	46.58	9.31	1.29	0.28
Within	26	187.63	7.21		
Total	31	234.21			

Table 7

Analysis of Variance Table

(using volume measurements of
the mesial aspect of tooth #8
as the variable)

<u>VARIANCE</u> <u>SOURCE</u>	<u>DF</u>	<u>SUM OF SQUARES</u>	<u>MEAN SQUARES</u>	<u>F RATIO</u>	<u>F PROB.</u>
Between	5	52.07	10.41	1.91	0.12
Within	26	141.42	5.43		
Total	31	193.50			

Table 8

Analysis of Variance Table

(using TOTAL volume measurements
of all three teeth as the variable)

<u>VARIANCE SOURCE</u>	<u>DF</u>	<u>SUM OF SQUARES</u>	<u>MEAN SQUARES</u>	<u>F RATIO</u>	<u>F PROB.</u>
Between	5	1,811.34	362.26	2.80	0.03
Within	26	3,358.12	129.15		
Total	31	5,169.46			

Table 9

MEAN VALUES OF POCKET DEPTH AND GCF METER READINGS

<u>Age Groups</u>	<u>Pocket Depth (mm)</u>	<u>GCF Meter Reading</u>
(6-12)	2.19	6.90
(13-20)	2.23	7.00
(21-30)	2.30	4.83
(31-45)	2.40	3.93
(46-60)	2.16	6.93
(60- +)	2.06	7.16
Average	2.22	6.13

DISTRIBUTION OF MALES AND FEMALES

<u>Age Groups</u>	<u>Males</u>	<u>Females</u>	<u>Total</u>
(6-12)	5	2	7
(13-20)	1	4	5
(21-30)	4	3	7
(31-45)	2	3	5
(46-60)	1	4	5
(60- +)	1	2	3
Total	14	18	32

Table 10

Table 11

THE COEFFICIENT OF CORRELATION

$$r = \frac{2592 - \frac{(430)(1158)}{192}}{\sqrt{\left(1002 - \frac{(430)^2}{192}\right) \left(8416 - \frac{(1158)^2}{192}\right)}} = -.006$$

$$r = -.006$$

TESTING THE SIGNIFICANCE OF THE COEFFICIENT OF CORRELATION

$$t = \sqrt{\frac{(-.006)^2 (190)}{1 - (-.006)^2}} = .0827$$

Null Hypothesis = Pocket Depth and Gingival Fluid Volume are Independent.

$t < 1.97$, therefore the null hypothesis can't be rejected.

CHAPTER V

DISCUSSION

Gingival fluid may be an inflammatory exudate or a altered serum transudate, but none-the-less, the flow rate is a extremely sensitive measure to evaluate the condition of the gingivae. The Periotron[®] has been shown to be valuable in assessing the condition of the gingival tissues (Golub & Kleinberg 1976; Suppipat 1976; Shern 1974; Borden 1976).

Gingival inflammation is the host's response to defend itself from bacterial invasion and its products entering the gingival crevice by diluting, removing or inactivating the foreign substance. Histological changes observed during inflammation are as follows:

1. PMN accumulation.

2. Vasodilation of arterioles, metarterioles and venules.

3. Increased capillary permeability allowing fluid loss.

This fluid enters the gingival sulcus before any clinical signs of inflammation occur.

Gingival crevicular flow has been shown to increase with the increase of gingival inflammation (Brill & Bjorn 1959; Mann 1963; Egelberg 1964; Oliver et al. 1969; Golub et al. 1971) and there is

a rise in the GCF flow prior to the appearance of any clinical signs of inflammation (Löe & Holm-Pedersen 1965). Therefore, if the possibility exists of identifying the increase of GCF flow before the clinical signs of inflammation occur, then the appropriate treatment could be administered before the inflammatory condition becomes more serious (chronic - degradation of the integrity of the periodontium decreasing the capacity and function of the stomatognathic system).

To evaluate the individual case properly, we need mean values of GCF flow for different age groups, sex, and tooth positions in the arch.

The present findings indicate there is no difference between GCF flow and sex, but there is a difference between GCF flow and age groups ($P < .05$). With these mean values of GCF flow for certain age groups and tooth position in the arch, a clinician can evaluate the gingival status of a patient with clinically normal gingivae and act accordingly to prevent subsequent periodontal destruction. Also, monitoring GCF flow can be used to:

1. quantify the severity of gingival inflammation,
2. evaluate the response of the gingivae to periodontal therapy,
3. evaluate oral hygiene procedures,
4. assist in the education of the patient with the use of simple numerical-severity relationship.

There has been and still is controversy on whether or not crevicular fluid flow is present in clinically healthy gingivae. The present investigation indicated there is a GCF flow in clinically healthy gingivae ($P < .001$). This flow could be a normal physiologic state yielding an altered transudate of serum (in agreement with Weinstein et al. 1967; L  e 1961). Only six pockets out of one hundred ninety two pockets measured exhibited no GCF flow.

Some investigators have determined a significant positive correlation to exist between GCF flow and pocket depth. The present investigation indicated no correlation ($r = -.006$) between GCF flow and pocket depth in individuals who exhibit clinically healthy gingivae. The distinction of clinically healthy gingivae in the previous statement is extremely important. A positive correlation may very well exist in individuals who exhibit degrees of inflammation, but when dealing with individuals exhibiting clinically healthy gingivae, there is no correlation between GCF flow and pocket depth.

Possible errors in relating mean GCF values determined in this study to a particular patient is as follows:

1. Technician variability

- A. Failure to isolate and dry area to be examined properly.

- Drying too much could irritate the tissue and influence GCF production.

- B. Failure to empty existing crevicular pool thoroughly.
 - C. Improper procedure holding filter paper strips in sulcus, using intracrevicular technique, for three seconds.
 - D. Improper placement of filter paper strips in the jaws of the periotron[®] (too far or not far enough).
2. Climate
- A. Temperature
 - B. Relative Humidity

It must be stressed measuring gingival crevicular fluid flow should not be used solely to evaluate the gingival tissue of a patient. It's true utilization of the Periotron[®] (Harco Electronics, Winnipeg, Canada) is sensitive and objective in evaluating the quality of the gingiva, but due to the possible errors in the sampling procedure the Periotron[®] shouldn't be used solely. To reduce the possibility of error, it is more meaningful to average several measurements within the same individual than to simply take a single measurement. The other important criteria to look at when evaluating gingival tissue is the Gingival Index (Löe and Silness, 1963). Monitoring GCF flow along with clinical examination utilizing the Gingival Index will prove to be a sensitive, quantitative, and practical means to evaluate the gingival status.

With the standardized gingival crevicular fluid meter values determined in this study, further investigations could be executed.

A few suggestions are as follows:

1. Determination of the effect of any restorative procedure.
2. Determination of the effect of different types of restorative material.
3. Determination of the value of periodontal treatment or surgery.
4. Determination of the effect of orthodontia on the periodontium.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The mean gingival crevicular fluid volume was determined for the following age groups: (6-12), (13-20), (21-30), (31-45), (46-60), and (60+). Thirty-two patients with clinically healthy gingivae (Gingival Index = 0; Loe and Silness, 1963) were selected for this study. The teeth measured in this study included maxillary right canine #6, lateral incisor #7, and central incisor #8. The crevicular fluid was collected intracrevicularly with the use of sterile filter paper strips and the volume was determined utilizing a Periotron[®] (Gingival Crevicular Fluid Meter, Harco Electronics, Winnipeg, Canada). In all pockets measured for GCF flow, the pocket depth was measured utilizing a perioprobe.

By evaluating the results of the present study, the following conclusions can be made:

1. In patients exhibiting clinically healthy gingivae, there is a significant difference between the means of GCF flow in different age groups ($P < .05$).
2. In patients exhibiting clinically healthy gingivae, there is no correlation between pocket depth and GCF flow.
3. There is a GCF flow in clinically healthy gingivae ($P < .001$).
4. In relation to GCF flow, there is no significant

difference between males and females.

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APPENDIX

LOYOLA DENTAL SCHOOL
Oral Biology Department
Crevicular Fluid Volume Measurements

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PATIENT CONSENT FORM

The purpose of this study is to determine the average amount of fluid flowing from the space between the teeth and gums.

This preliminary study will utilize an instrument that measures very small amounts of fluid and possibly can be used to detect gum disease before it has progressed to an advanced stage.

The procedure will involve isolating the area with a sterile cotton roll and then placing a small sterile piece of filter paper next to the teeth near the gums for about three (3) seconds. Several teeth will be measured in this manner. This will not produce any discomfort or have any ill effect whatsoever on the gums or teeth. The entire procedure including filling out the questionnaire should take approximately twenty (20) minutes.

If at any time during the procedure you want to withdraw your participation in this study you are free to do so.

I HAVE READ THE ABOVE INFORMATION AND WILL PARTICIPATE IN THIS STUDY.

PARTICIPANT'S SIGNATURE _____

DATE _____

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If yes, how much? _____

Clinical Examination and Evaluation

Deposits - Stain ☐ L ☐ M ☐ H Type _____ 70

Supragingival Calculus ☐ L ☐ M ☐ H

Subgingival Calculus ☐ L ☐ M ☐ H

Gingivae ☐ Normal ☐ Inflamed-Localized ☐ Inflamed-Generalized
☐ Recession

Soft Tissue lesions: ☐ Absent ☐ Present-Location _____

Teeth: Caries ☐ Absent ☐ Present-Insipient ☐ Present-Rampent

☐ Decalcification ☐ Attrition ☐ Abrasion ☐ Erosion

Missing - ☐ Max. Ant. ☐ Mand. Ant.

☐ Max. Post. ☐ Mand. Post.

Mobility - ☐ Normal ☐ Excessive in one or more teeth

Occlusion

First Molar - ☐ Cl. I ☐ Cl. II ☐ Cl. III

Angle's - ☐ Cl. I ☐ Cl. II - Div. I ☐ Cl. II - Div. II

☐ Cl. III ☐ Overbite ☐ Overjet ☐ Ant. openbite

Open Contacts ☐ Present ☐ Absent

Facies - ☐ Dolichocephalic (Long and Narrow)

☐ Brachycephalic (Short and Broad)

Skeletal Pattern ☐ Ectomorph (Slender-Fragile-Cerebrotonic)

☐ Mesomorph (Strong-Muscular-Somatotonic)

☐ Endomorph (Soft-Round-Viscerotonic)

Patient _____ Operator _____
 Address _____ City _____ State _____
 Phone # _____ Age _____ Birth Date _____ ZIP _____
 Sex: ____M ____F

TOOTH #	Mobility	Poc't Depth (mm)	Gingival Index	Volume
6 distal				
6 medial				
7 distal				
7 medial				
8 distal				
8 medial				

Note:

Mobility: Use Arbitrary
Scale Below:

Rating from 0 to 3

- a. 0 = No perceptible
Mobility
- b. 3 = Hopeless prognosis
due to extreme
mobility
- c. D-III = Tooth is
Depressible in Socket

Gingival Index (After Loe & Silness-
J. Perio. 38:610, 1967)

- a. 0 = Normal gingiva
- b. 1 = Mild inflammation, slight
change in color, slight
edema, no bleeding on
probing.
- c. 2 = Moderate inflammation,
redness, edema, and glazing;
bleeding on probing.
- d. 3 = Severe inflammation, marked
redness and edema; ulceration

APPROVAL SHEET

The thesis submitted by Gary William Brankin has been read and approved by the following committee:

Dr. William F. Malone, Director
Professor, Fixed Prosthodontics, Loyola

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Professor & Chairman of Oral Pathology, Loyola

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The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the thesis is now given final approval by the Committee with reference to content and form.

The thesis is therefore accepted in partial fulfillment of the requirements for the degree of Master of Science.

Date

5/9/77

Director's Signature

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